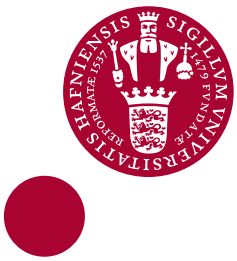

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## science behind the myth

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# Physical activity, sleep duration and metabolic health in children fluctuate with the lunar cycle: science behind the myth

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## Summary

Behaviours of several animal species have been linked to lunar periodicity. Evidence for such links in humans is weak; however, recently, shorter sleep duration was reported around full moon in two small samples of adults. As restrictions in sleep duration have been shown to adversely affect glucose regulation and physical activity to improve glucose regulation, one could speculate that cardiometabolic risk factors might also be affected by the lunar phase. We retrospectively examined 795 Danish children, aged 8–11 years, with more than 13 000 24-h accelerometer recordings of activity and sleep as well as 2000 measurements of different cardiometabolic risk factors, including insulin sensitivity, appetite hormones and blood pressure, during nine lunar phases. During the period around full moon, children were 5.0 and 3.2 min per day less active, slept 2.4 and 4.1 min per night longer, had 0.03 and 0.05 higher homeostatic model assessment of insulin resistance and 0.6 and 0.8 mmHg higher mean arterial blood pressure compared with days around half moon and new moon, respectively (all $P \leq 0.02$). Furthermore, ghrelin was lower and leptin was higher during the period around full moon compared with days around half moon (both $P < 0.001$). The results suggest that physical activity rather than sleep is responsible for the metabolic alterations observed around full moon. However, we have no understanding of potential mechanisms that may mediate a potential true link between childhood behaviour and the lunar cycle or confounders that may explain this, apparently leading to fluctuation in a number of cardiometabolic risk markers conjointly with lunar phases.

**Keywords:** Insulin, moon, physical activity, sleep.

## Introduction

The term 'lunacy', derived from Luna, the Roman goddess of the moon, has been used since ancient times to explain behaviour and mental health. Folklore and even certain instances of occupational lore suggest that the behaviours of both humans and animals, including werewolves, are affected by lunar phases. In particular, the period around full moon has been associated with increased prevalence of sleep problems, seizures, cardiovascular events, anti-social behaviour and mental problems (1). Behaviours of several animal species have been linked to lunar periodicity (2); the phenomenon of the Palolo worms, which reproduce by mass spawning during the last quarter of the moon, is one of the best-known examples (3). Currently, the scientific bases for such links in humans are weak. It has, however, recently been reported that sleep duration was negatively affected around full moon in two small samples of adults (4,5). As restrictions in sleep duration have been shown to adversely affect glucose regulation (6), one could speculate



 

that cardiometabolic risk factors because of insufficient amounts of quality sleep might be affected by the lunar phase. Furthermore, physical activity has the potential to improve glucose regulation (7) and, in addition to sleep, could explain potential differences in metabolic health during the lunar phases.

To test this myth, we retrospectively examined 795 Danish children, aged 8–11 years, having accelerometer-determined sleep duration and daytime physical activity behaviour. We collected data from a total of 13 762 nights and 13 464 d and made more than 2000 measurements of different cardiometabolic risk factors, including insulin sensitivity, appetite hormones, inflammation, blood lipids and blood pressure, in relation to the lunar phase.

## Materials and methods

The initial sample comprised 834 of the 1021 invited third- and fourth-grade students (8–11 years old) from nine Danish municipal schools enrolled in the OPUS (Optimal well-being, development and health for Danish children through a healthy New Nordic Diet) school meal study. The main aim of this cluster-randomized crossover study was to investigate the potential health effects of a New Nordic Diet served at school vs. the usual packed lunch (control) (8). Measurements were performed at baseline (August to November 2011), before the end of the first dietary period (approximately 100 d later) and before the end of the second dietary period (after another 100 d). As the school meal intervention did not affect physical activity, sedentary time and sleep duration (9), all three measurement points were used in the present paper regardless of randomization status. The present analytical sample comprised data from the 795 children having valid sleep, daytime physical activity or cardiometabolic risk factors from at least one of the three measurement periods as well as a valid reporting of pubertal status at baseline resulting in 13 464 observations of daytime activity, 13 762 observations of sleep duration and 2005 to 2148 observations of cardiometabolic risk factors across nine moon cycles from August to June. The study was approved by the Committees on Biomedical Research Ethics for the Capital Region of Denmark (J.nr. H-1-2010-123). Child assent and written informed parental consent of both custody holders were obtained for all participants. The study was registered in the www.clinicaltrials.gov database (no. NCT01457794).

### Daytime activity and sleep duration

The children were asked to wear an ActiGraph™ tri-axis accelerometer monitor (GT3X+ or GT3X, Pensacola, FL, USA) tightly on the right hip using an elastic belt continuously for seven consecutive days and eight nights; they were only allowed to remove it during water activities (i.e. showering or swimming). At the end of the observation period, data were reintegrated to 60-s epochs and analysed using ActiLife6 (ActiGraph, Pensacola, FL, USA). Before analysis of physical activity and sedentary time, we removed: (i) data between midnight and 6.00 a.m., as this was expected to be non-awake time; (ii) periods of at least 15 min of consecutive zero counts using tri-axial vector magnitudes to remove non-wear time and non-awake time; and (iii) consecutive wear time periods of less than 60 min to remove non-awake time, as sleep for most children is characterized by minor periods of movement that we did not want to include in our analysis of daytime activity. Sedentary time, light physical activity and moderate-to-vigorous physical activity were defined as all minutes showing ≤100 vertical cpm, 101–2295 vertical cpm and ≥2296 vertical cpm, respectively; these are widely used cut-off points (10). Daytime activity was only considered valid if monitor wear time was at least 10 h per day.

The parents and children were instructed to keep logs for bedtime ('lights off' and trying to sleep) and waking time ('lights on') during the week in which the monitor was worn. To estimate accelerometer-determined sleep duration, the self-reported bedtimes and waking times were used as the possible window of sleep and accelerometer data within this window were scored in ActiLife6 using the algorithm by Sadeh *et al*. that counts minutes below a certain activity threshold as sleep (11). This algorithm was developed using wrist-worn monitors; however, we recently tested the agreement between wrist and waist-worn ActiGraph monitors using this algorithm and concluded that waist-worn devices can provide a valid proxy measure of sleep duration in epidemiological studies (12).

### Anthropometry and cardiometabolic risk markers

Clinical measurements and venous blood sampling from the antecubital vein were performed in the morning (time interval: 7.25–10.45 a.m.) after an overnight fast in a mobile laboratory. Whole blood glucose was analysed immediately after sampling using a Hemocue Glucose 201 analyser (Hemocue Danmark, Brønshøj, Denmark) that calculated plasma glucose concentrations from whole blood concentrations. Heparinized blood and blood with ethylenediaminetetraacetic acid were centrifuged at 2500 g for 10 min at room temperature and aliquoted plasma was stored at −80°C. Serum insulin, plasma triglycerides, plasma cholesterols, plasma leptin and plasma ghrelin were analysed according to standard protocols described elsewhere (13,14). Plasma C-reactive protein (CRP; high sensitive) was quantified on the Vitros 5.1 FS analyser (Ortho–Clinical Diagnostics, Johnson & Johnson, Birkerød, Denmark) with a lower CRP detection limit of 0.1 mg $L^{-1}$. Plasma interleukin (IL)-6 (high sensitive) and adiponectin were measured in duplicate by enzyme-linked immune-

 

sorbent assays (R&D Systems Europe Ltd., Abingdon, UK). Homeostatic model assessment of insulin resistance ($HOMA_{IR}$) was calculated as plasma glucose (mmol $L^{-1}$) × serum insulin (mmol $L^{-1}$)/22.5 (15). The inter- and intra-assay coefficients of variation were: 1.3% and 0.8% (CRP); 6.7% and 2.9% (IL-6); 9.2% and 3.7% (leptin); 11% and 3.8% (adiponectin); 9% and 3.7% (ghrelin); 1.8% and 1.2% (total cholesterol); and 1.9% and 1.2% (High-density lipoprotein cholesterol). The inter-assay coefficients of variation were 3.4% for glucose and 5.9% for insulin. After 10 min of rest, blood pressure and heart rate were measured three times in the supine position with an automated device (UA-787 Plus; A&D Medical, San Jose, CA, USA) using two different cuff sizes (18–22 cm or 22–32 cm). A second device (ProBP 3400 Sure BP, Welch Allyn Inc., New York, USA) was used for children with arm circumferences <22 cm using three different cuff sizes (12–16, 15–21 and 20–26 cm). The mean of the last two measurements was used in the data analyses. The mean arterial blood pressure was calculated as: 1/3 × systolic blood pressure (mmHg) + 2/3 × diastolic blood pressure (mmHg). Height (CMS Weighing Equipment LTD, London, UK) was measured three times to the nearest millimetre and the average was used. The children were weighed to the nearest 0.1 kg (Tanita BWB-800S, Tanita, Europe) while barefoot and wearing light clothing. The prevalence of underweight, normal-weight, overweight and obese children was calculated based on age- and sex-specific cut-offs defined to pass through a body mass index of 18.5, 25 and 30 kg $m^{-2}$ at 18 years of age (16,17). Total body fat was determined by dual-energy X-ray absorptiometry (Lunar Prodigy; GE Medical Systems, Madison, WI, USA) using Encore software version 13.5 (Encore, Madison, WI, USA).

### Questionnaire data

A baseline questionnaire ascertained age, gender, school grade, highest education of the parents (divided into four groups according to years of education: ≤10 years, 11–12 years, 13–16 years, ≥17 years) and number of parents born in Denmark (proxy of ethnicity). A proxy of pubertal status was self-reported (parent and child) based on breast development among girls and pubic hair growth among boys on a scale from 1 to 5 (18). A dichotomous variable indicating whether or not the child had entered puberty was used in the statistical analyses (1 or ≥2).

### Statistical analysis

Descriptive characteristics of the study sample were presented as mean and standard deviation, median (interquartile range) or as proportions. An available-case linear mixed model with school, class and subject as random effects and weekday, month, age, gender, Tanner stage, number of parents born in Denmark and highest education of parents as fixed effects was used to test lunar rhythmicity in behaviour (sleep, sedentary time, light physical activity and moderate-to-vigorous physical activity) and metabolic variables ($HOMA_{IR}$, glucose, insulin, blood pressure, ghrelin, leptin, CRP, IL-6, adiponectin, high-density cholesterol, low-density cholesterol, triglycerides and resting heart rate). Based on this model, we estimated the adjusted behaviour/biomarker with full moon ±4 d (full moon) as reference compared with ±5–9 d (half moon) and ±10–14 d (new moon) from nearest full moon. These classifications of lunar phases are identical to what have been used in recently published papers (4,5,19) using information from a full moon calendar (20). Estimates are presented as unstandardized regression coefficients (β) with 95% confidence intervals. Some metabolic variables were positively skewed and were thus log-transformed for analyses and back-transformed after analyses. Adjusting for the fact that the three repeated measures came from baseline, control and intervention did not affect the observed associations between metabolic variables and lunar phases. Presented *P*-values were not adjusted for multiple testing but significant associations vanishing after Bonferroni adjustment were indicated. Pearson's correlation coefficients were obtained between sleep duration, moderate-to-vigorous physical activity and $HOMA_{IR}$ using an average of the three measurements obtained at baseline, day 100 and day 200. Changes from summer to winter time (clock set back by 1 h) and from winter to summer time were excluded from data analysis. The level of significance was set at $P < 0.05$ and statistical analyses were conducted using STATA/IC 11.2 (Houston, TX, USA).

## Results

Baseline characteristics of the children can be found in Table 1. As shown in Table 2, accelerometer-determined

**Table 1** Descriptive characteristics of the study population at baseline ($n$ = 788–793)

| Variable | Mean ± standard deviation or proportions |
|---|---|
| Age (years) | 10.0 ± 0.6 |
| Gender (% boys) | 51.8 |
| Tanner stage (% 1/2/≥3) | 65.6/27.5/6.9 |
| Parents born in Denmark (% 0/1/2) | 8.7/12.2/79.1 |
| Highest education of parents (%)* | 5.4/34.5/38.8/21.2 |
| Body mass index status (% uw/nw/ow/ob)† | 10.3/76.4/11.4/1.9 |

Data are presented as mean ± standard deviation or proportion.
*Highest education of parents: ≤10/11–12/13–16/≥17 years.
†Based on age- and sex-specific cut-offs defined to pass through body mass index at 18.5, 25 and 30 kg $m^{-2}$ at age 18 years (16,17); uw/nw/ow/ob, underweight/normal weight/overweight/obese.

 

**Table 2** Behaviours and metabolic variables according to lunar phases ($n$ = 751–793)

| Variables | Observations | Mean ± standard deviation or median (interquartile range) | Full moon† – half moon‡ | Full moon† – new moon§ |
|---|---|---|---|---|
| Behaviours | | | | |
| Moderate-to-vigorous PA (min per day) | 13 464 | 41 (21; 68) | –5.0 (–6.5; –3.6)** | –3.2 (–4.7; –1.7)** |
| Light PA (min per day) | 13 464 | 375 ± 80 | 0.1 (–3.0; 3.2) | 1.7 (–1.4; 4.9) |
| Sedentary time (min per day) | 13 464 | 477 ± 99 | 1.4 (–2.6; 5.4) | –1.9 (–6.0; 2.2) |
| Sleep duration (min per night) | 13 762 | 550 ± 49 | 2.4 (0.3; 4.4)* | 4.1 (2.0; 6.2)** |
| Metabolic variables | | | | |
| $HOMA_{IR}$ | 2005 | 1.46 (1.03; 2.05) | 0.03 (0.005; 0.05)* | 0.05 (0.02; 0.08)* |
| Insulin (pmol $L^{-1}$) | 2005 | 43.8 (31.6; 60.5) | 0.09 (0.004; 0.18)*†† | 0.15 (0.03; 0.27)* |
| Glucose (mmol $L^{-1}$) | 2057 | 5.2 ± 0.5 | 0.05 (0.005; 0.10)*†† | 0.12 (0.06; 0.18)** |
| Mean arterial blood pressure (mmHg) | 2148 | 79.5 ± 6.0 | 0.6 (0.1; 1.1)* | 0.8 (0.2; 1.4)* |
| Diastolic blood pressure (mmHg) | 2148 | 66.0 ± 6.4 | 0.7 (0.2; 1.2)* | 0.9 (0.3; 1.6)* |
| Systolic blood pressure (mmHg) | 2148 | 106.5 ± 7.6 | 0.4 (–0.2; 0.9) | 0.4 (–0.3; 1.2) |
| Ghrelin (pg $mL^{-1}$) | 2028 | 996 ± 383 | –29 (–46; –13)** | –5 (–28; 18) |
| Leptin (pg $mL^{-1}$)¶ | 2017 | 4013 (2223; 8056) | 1.2 (0.5; 1.8)** | 0.7 (–0.1; 1.6) |

Data are presented as unstandardized regression coefficients (β) or % difference with 95% confidence intervals using a linear mixed model with school, class and subject as random effects and weekday, month, age, gender, Tanner stage, number of parents born in Denmark and highest education of parents as fixed factors.

†Full moon ±4 d (Include 25 to 35% of the observations in all models).

‡±5–9 d from full moon (Include 35 to 36% of the observations in all models).

§±10–14 d from full moon (Include 29 to 39% of the observations in all models).

¶Additionally adjusted for total body fat.

††Significant associations vanished after Bonferroni adjustments.

*$P < 0.05$; **$P < 0.001$ indicate significant difference from full moon ±4 d.

$HOMA_{IR}$, homeostatic model assessment of insulin resistance; PA, physical activity.

moderate-to-vigorous physical activity was 5.0 and 3.2 min per day lower (both $P < 0.001$) and sleep duration was 2.4 and 4.1 min per night longer (all $P \leq 0.02$) at full moon compared with half moon and new moon, respectively. Cardiometabolic risk markers as $HOMA_{IR}$, insulin, glucose, mean arterial blood pressure and diastolic blood pressure were all higher at full moon compared with half moon and new moon (all $P \leq 0.04$); $HOMA_{IR}$ was 0.05 higher at full moon compared with new moon. Furthermore, leptin was 1.2 pg $mL^{-1}$ higher and ghrelin 29 pg $mL^{-1}$ lower at full moon compared with half moon (both $P < 0.001$). No differences according to lunar phase were found for time spent sedentary, light physical activity, CRP, IL-6, adiponectin, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, triglycerides or resting heart rate (all $P > 0.15$). In our investigation of plausible connections between behaviours and metabolic health, we tested and found a negative correlation between moderate-to-vigorous physical activity and sleep duration ($r = –0.12$, $P = 0.01$); between moderate-to-vigorous physical activity and $HOMA_{IR}$ ($r = –0.25$; $P < 0.001$); and between sleep duration and $HOMA_{IR}$ ($r = –0.16$; $P < 0.001$) after adjustment of sleep duration and moderate-to-vigorous physical activity, respectively. Bonferroni adjustment resulted in non-significant differences in insulin ($P = 0.08$) and glucose ($P = 0.06$) between full moon and half moon; however, all other significant associations remained.

## Discussion

This is the first study to demonstrate that variations in sleep duration as well as in daytime physical activity are associated with the lunar circle (~29.5 d) and, furthermore, that a number of cardiometabolic risk markers fluctuated in accordance with this. Contrary to what has been hypothesized and recently shown in adults (4,5), children slept somewhat longer around full moon. It should be noted that, although this was statistically highly significant, the fluctuation in sleep duration between lunar phases was small and its biological relevance is questionable. However, we found that children were also less physically active during this period and furthermore that this coincided with biologically meaningful differences in several cardiometabolic risk factors, including lower insulin sensitivity and higher blood pressure, while the blood lipid profile, adiponectin and markers of inflammation were not affected. In addition, we found lunar phase-associated variations in ghrelin and leptin in directions that could favour a positive energy balance during the period around full moon (21).

The negative correlation between moderate-to-vigorous physical activity and sleep duration fits well with the observation that sleep is longest and activity lowest during full moon. However, the differences in sleep duration were very small compared with those differences seen for moderate-to-vigorous physical activity and the negative correlation

 

observed between sleep duration and $HOMA_{IR}$ does not fit with $HOMA_{IR}$ being highest during full moon. We therefore speculate that any potential physiological effect of the lunar phase is not mediated by an effect on sleep, but may involve alterations in physical activity. These changes in daytime activity could then subsequently alter sleep without a direct effect of the lunar cycle.

While potential effects of the moon on daytime activity and cardiometabolic risk factors have never been studied in humans, an effect on sleep has been shown in three previous studies. Based on sleep logs from 31 adults over 6 weeks, Röösli *et al*. were the first to demonstrate that humans slept less (19 min) at full moon compared with at new moon (22). This finding was recently confirmed by Cajochen *et al*. (4) as well as Smith *et al*. (5), who found differences of 20 and 25 min using objective sleep measures obtained from electroencephalography (EEG) monitoring under confined laboratory conditions in 33 adults measured for two nights and 47 adults measured for six consecutive nights, respectively. Other researchers have investigated this without finding an influence of the lunar cycle on human sleep (19,23–25). Binkley *et al*. reported no difference in self-reported sleep between full moon and new moon in four adults followed for 1 year (24); Pandey *et al*. found no correlation between fraction of the moon illuminated and self-reported sleep in 43 adults followed for 105 d (25); and an abstract by Zeitlhofer *et al*. did not find subjective ratings of sleep at full (189 nights), new (152 nights), waxing (176 nights) and waning (174 nights) moons to differ in 391 adults followed for 14 consecutive nights (23). Finally, Cordi *et al*. recently presented three separate analyses consisting of 470, 757 and 870 nights of EEG sleep recordings of adults which were unable to support previous findings on sleep (19).

In the study by Cajochen *et al*., subjects were allowed to sleep *ad libitum* without external time queues, while children in our study were assessed over a whole week, when they were likely woken at a fixed time on weekdays in order to attend school. This to some extent may have defined their sleep duration and limited our ability to find effects on sleep duration. However, it cannot explain the small but quite consistent longer sleep around full moon. In addition to shorter total sleep duration around full moon, Cajochen *et al*. also reported longer rapid eye movement sleep latency, longer sleep latency, lower subjective sleep quality and lower levels of evening melatonin during this period (4). In support, Smith *et al*. reported longer sleep onset latency, but also a shorter REM sleep latency around full moon (5). Although accelerometers offer an objective method to quantify sleep duration, it cannot detect the different sleep stages. Therefore, we cannot rule out that the longer sleep duration of the children around full moon is a consequence of poorer sleep quality. However, as sleep duration was measured by counting minutes below a certain activity threshold, it is unlikely that the longer sleep duration was caused by awakenings during the night.

The lower levels of evening melatonin, shorter sleep duration and poorer sleep quality around full moon observed by Cajochen *et al*. fits with the well-established positive association between sleep and melatonin (26). Recent studies indicate that low levels of melatonin are associated with a higher risk of developing type 2 diabetes (27), providing a link between insufficient sleep and metabolic health and further indicating that the participants in the study by Cajochen *et al*. (4) could also have been metabolically affected by the behavioural changes observed in response to the lunar cycle. We observed highly meaningful physiological differences in cardiometabolic risk markers, including glucose regulation, which fits well with the fluctuations observed in moderate-to-vigorous physical activity (7).

Using more than 13 000 d and nights of objectively measured behaviour and more than 2000 measurements of different cardiometabolic risk factors across nine lunar cycles, this paper offers evidence in support of an effect of lunar rhythmicity on human behaviour and metabolic health. On average, each child had valid activity and sleep recordings from 17 d and 18 nights used in the analyses. Further, we adjusted for a large number of potential confounders shown to affect physical activity and sleep duration in this group of children (9). Finally, the linear mixed model approach removes differences between children before estimates are calculated. Therefore, it is very unlikely that our findings could be explained by other sources of variation (e.g. uneven distribution of age and gender), as might have been the case in the study by Cajochen *et al*., where there were almost three times as many older subjects around full moon compared with new moon (4). However, we lack strong hypotheses regarding plausible mechanisms that can explain the association between the lunar phases and variation in daytime activity and/or sleep. Several publications on the various influences of lunar phases discuss the potential role of variations in gravitational forces that, e.g., in some areas of the world have considerable effects on water, seen as tides. The tides are affected by the combined gravitational forces exerted by the moon and the sun as well as the rotation of the earth, which result in rises and falls of water levels every 12.4 h. Spring tides (higher than normal tides) and neap tides (high tides are lower than normal and low tides are higher than normal) are showing semi-lunar cycles (~14.8 d) with spring tides peaking at full and new moon and neap tides peaking at half moon (28). However, as we find large differences in behaviour and metabolic risk markers between full moon ±4 d and ±10–14 d from full moon, periods that both coincide with spring tides, these gravitational forces cannot explain our findings. Further, these forces are said to be remarkably weak (1).

 

In the same group of children, we have previously reported shorter sleep duration during periods of the year with longer photoperiods (day length) (9), which leads us to yet another proposed mechanism involving differences in light exposure during the night, which is known to affect sleep (29). At full moon during a clear night, illuminance may be around 25 times greater than at half moon and up to 250 times greater compared with a moonless night. In modern urban societies where most of us are surrounded by an abundance of artificial light and spend evenings and nights mostly indoors, this effect is probably minor (1) and light in the late evening and at night is unlikely to have an effect on physical activity during the time awake.

The results suggest that physical activity rather than sleep is responsible for the metabolic alterations observed around full moon. However, at the moment, we have no understanding of potential mechanisms that may mediate a potential true link between childhood behaviour and the lunar cycle or of confounders that may provide alternative explanations for the associations we found between lunar phases and behaviour apparently leading to fluctuation in a number of cardiometabolic risk markers conjointly with lunar phases. Solving the lunar mystery could lead to novel, fascinating and unexpected insight into the effects of the environment on human behaviour and our physiology in general.

## Conflict of Interest Statement

No conflict of interest was declared.

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